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Ascending aortic obstruction produced by dissected intimal flap*

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SUMMARY A case of aortic dissection is presented in which the patient developed chest pain, syncope, and new heart murmurs. An aortic supravalvular gradient of 39 mmHg was found across a thickened dissected intimal flap which partially occluded the ascending aorta. The recognition of this form of aortic occlusion may be important in the clinical evaluation of patients with aortic dissection.

The following case report illustrates that supravalvular aortic obstruction may be produced by a dissected intimal flap in the proximal ascending aorta. This mechanism of aortic obstruction has not been previously described.

Case report

The patient, a 67-year-old woman, mildly hypertensive for 13 years, experienced an episode of severe interscapular back pain associated with syncope two years previously. She had been admitted to hospital for six weeks and told she had had a "heart attack". Two weeks before admission she had another episode of severe interscapular back pain, with radiation down both arms, again associated with syncope of unknown duration. She did not seek medical attention for three days after this episode during which she became increasingly dyspnoeic on exertion. When seen by her doctor prominent systolic and diastolic murmurs were heard, apparently for the first time, and she was referred to hospital.

On physical examination there were no stigmata of Marfan's syndrome. The carotid arteries showed a brisk upstroke and there were bruits on both sides. A prominent sustained apical impulse was noted in the sixth intercostal space in the anterior axillary line, with an additional ectopic impulse along the left sternal border in the second intercostal space. Pulsation of the left sternoclavicular joint was present. A systolic ejection murmur, grade 3/6, was present, loudest in the third left intercostal space and radiating to the carotids. A diastolic decrescendo murmur grade 3/6 was also heard, loudest in the right fourth and fifth intercostal spaces. Neurological examination was normal.

The electrocardiogram showed sinus rhythm with R wave voltage and ST and T wave changes compatible with left ventricular hypertrophy. Left ventricular enlargement and an aneurysmal dilatation of the ascending aorta were present on *x*-ray films of the chest.

Cardiac catheterisation was performed from the right brachial artery and a right antecubital vein; the left brachial artery was cannulated for pressure monitoring. The pressure measurements are given in the Table.

Table

	Pressures (mmHg)	
	Phasic	*Mean*
Right brachial	122/45	96
Left brachial	122/45	96
Left ventricle	161/10	
Proximal ascending aorta	161/45	121
Distal ascending aorta	122/45	96

Cardiac output (dye dilution): 1·45 l/min per m².

On angiography left ventricular wall motion was normal, without defects. The left ventricular fraction was 59 per cent. A supravalvular injection of radiographic contrast material disclosed severe aortic regurgitation. A dissection of the proximal ascending aorta was present with the production of a large aortic aneurysm in the proximal portion of the ascending aorta. A large intimal flap extended across the aortic lumen from the anterior to the

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posterior wall (Fig. 1). As the catheter was withdrawn across the intimal flap a peak to peak gradient of 39 mmHg was recorded (Fig. 2). The false channel opacified readily with contrast material.

At operation, two days later, an entrance intimal tear was noted 2 cm above the aortic valve. The dissection extended to a point 1 cm proximal to the origin of the innominate artery. The dissected intimal flap was noted to be severely thickened. The aneurysm was resected and replaced with a 30 mm woven Dacron graft. The aortic valve appeared to be intrinsically competent and thus was not replaced.

After operation, she developed a severe coagulopathy, unresponsive to replacement with fresh frozen plasma, platelets, cryoprecipitate, or amino caproic acid, continued to bleed, and died six hours after operation.

Pathological examination of the resected aorta tissue showed severe fibrous thickening of the intima and adventitia, with a severely thinned and fragmented media. Changes in the media consistent with cystic medial necrosis were observed. Neointima formation of the false channel was noted, suggesting the dissection was chronic.

Discussion

Dissection of the aorta may produce branch vessel occlusion or partial obstruction in approximately 50 per cent of cases.[1] Frequently, extension of the dissection into the branch vessels occurs, with or without distal re-entry. Alternatively an intimal tear may occur at the vessel orifice, or the intima may become completely detached with separation of the branch vessel from the true lumen. Finally, branch vessel occlusion may occur as the orifice is compressed by a large false channel.[2] Obstruction of the ascending aorta by a large dissected intimal flap with a ball-valve action has not been previously

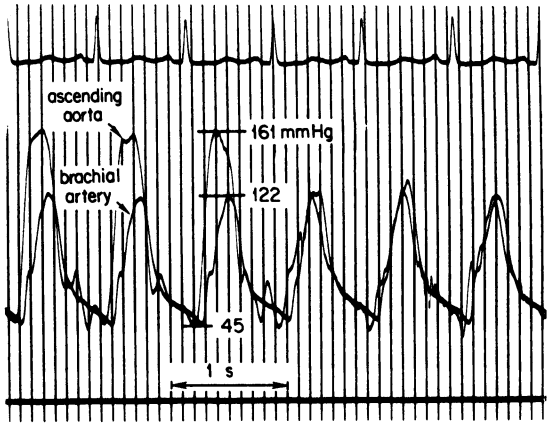

Fig. 2 *Simultaneous pressure recordings from the left brachial artery and the ascending aorta, proximal to the ascending intimal flap, demonstrate a 39 mmHg gradient. The gradient was confirmed as the aortic catheter was withdrawn across the flap.*

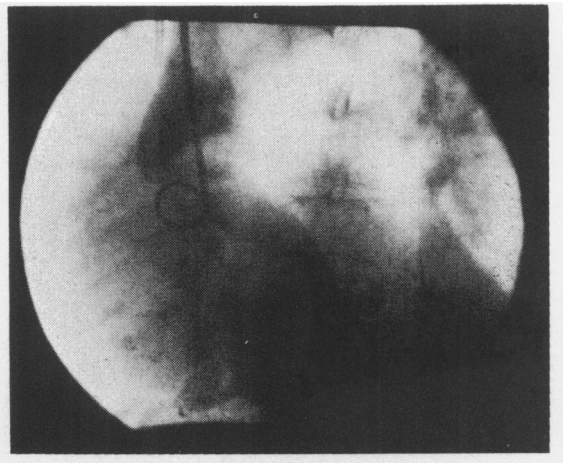

Fig. 1 *A large ascending aortic aneurysm is apparent in this left anterior oblique projection. The false channel opacifies readily and a large occlusive intimal flap extends from the anterior to the posterior aortic wall.*

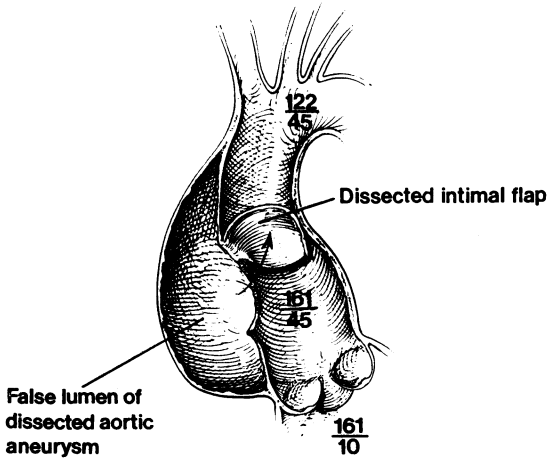

Fig. 3 *A large thickened dissected intimal flap was found at surgery. The thickened fibrotic character of the flap, as well as its size, contributed to a ball-valve effect, with the resultant ascending aortic obstruction.*

described. In the present case, the ability of such a flap to obstruct the aorta was attributable both to its size and to its thickened, fibrotic character. The patient's history, haemodynamic and pathological findings suggest that the dissection was chronic, with a recent extension approximately two weeks before admission

In the clinical evaluation of patients with aortic dissection, inequality of the limb pulses is strong evidence for direct involvement of a branch vessel by the dissection,[3] the mechanisms of which have been enumerated above. This case illustrates an additional, previously unreported, potential mechanism by which a dissected aortic intimal flap may partially occlude the ascending aorta.

Our patient also described syncope with her two episodes of severe interscapular pain. In a large series of patients with aortic dissection, syncope with subsequent recovery occurred in 6 to 13 per cent of cases.[3] Temporary occlusion of the orifices of the brachiocephalic vessels,[4] pain-induced vasodepressor reflexes, and complete atrioventricular heart block secondary to haematoma formation in the interatrial septum[5] have been described as causes of transitory alterations in consciousness, including syncope. In the present case, it is possible that a temporary increase in aortic obstruction producing vertebro-basilar insufficiency was the cause.

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